Richard P. Cambria, MD, Section Editor

Options for treatment of spontaneous mesenteric artery dissection

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Objective: **Mesenteric artery dissection was once thought to be rare but has been identified more frequently with increasing use of computerized tomographic angiographic imaging. Multiple reports advocate a wide range of treatment including medical therapy, endovascular, and surgical intervention, with no clear guidelines for the application of each treatment.**
Methods: **A systematic review of the literature was analyzed and used to create a treatment algorithm that was applied to nine patients in our institution.**
Results: **Although medical therapy is adequate for most patients with mesenteric artery dissection, 14% failed medical therapy, sometimes with severe consequences. Asymptomatic status on presentation predicted successful medical therapy for superior mesenteric artery dissection ($P = .0037$).**
Conclusions: **Intensive surveillance is indicated during medical therapy, and invasive intervention may be necessary. (J Vasc Surg 2014;59:1433-9.)**

Mesenteric artery dissection (MAD) was once thought to be rare but has been identified more frequently with increasing use of computerized tomographic arterial (CTA) imaging. The etiology of MAD not associated with trauma or iatrogenic causes remains unclear. Although aortic dissection has been associated with abnormalities of the aortic wall such as atherosclerosis, cystic medial necrosis, fibromuscular dysplasia, or connective tissue abnormalities (Marfan and Ehlers-Danlos syndrome), no such predisposing factors have been identified with MAD. Hypertension has been identified in at least 66% of patients with aortic dissection but only 31% of patients with MAD.[1] In a limited number of pathological specimens available, no histologic abnormalities other than the dissection were identified. Park has observed from a review of CTA scans in 51 patients that superior mesenteric artery (SMA) dissection generally begins at the anterior wall of the SMA near the convex curvature. With the use of a computer simulation model, abnormal hemodynamic factors were consistently observed at this site, suggesting mechanical stress as the etiology for SMA dissection.[1] No such investigation has been performed for celiac dissections.

MAD treatment was originally limited to surgical bypass for symptomatic cases; asymptomatic cases probably went unrecognized. Recent options for medical therapy and endovascular stenting have become popular but confusing as to when each option is applicable. Confusion arises from the fact that all dissections are not created equal. Park has categorized SMA dissection into four types (Fig 1),[1] but these subgroups do not take into account the length of the dissection or involvement of major branches. Nevertheless, most asymptomatic patients have a patent dual lumen with good distal flow (type I), whereas symptomatic patients present with (1) compromised true lumen secondary to compression by the false lumen, which has no re-entry site (type II), or (2) complete occlusion of the SMA (type III). Finally, the adequacy of collateral circulation may vary among patients. Other more complex subdivisions of MAD anatomy do not correlate with symptoms or treatment.[2-4]

Most clinicians would assume that persistent abdominal pain in the setting of MAD is a result of bowel ischemia. Some patients treated medically, however, have had good outcomes with observation, with abdominal pain resolving over a 7- to 14-day period. Yet, other patients treated medically have failed observation, ultimately requiring surgical or endovascular intervention with bowel resection. Yun has suggested that pain may be caused by distention of periarterial nerve fibers independent of bowel ischemia. A direct correlation between severity of pain and length of dissection has been observed.[5] How does the physician determine which symptomatic patient can be observed and which requires intervention?

METHODS

PubMed was searched for all English language reports of celiac and SMA dissection from 2002 to 2013. The bibliography of each report was examined for additional studies. All case reports and series were included in the

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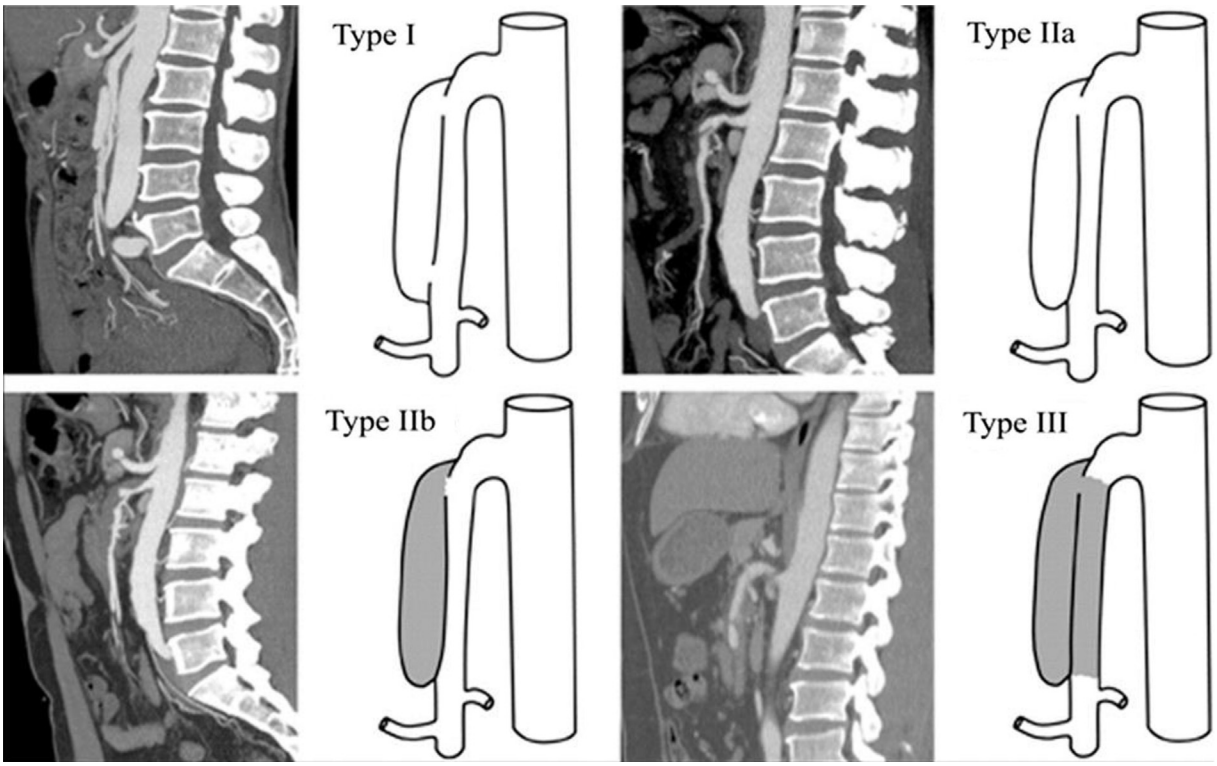

Fig 1. Angiographic classification of spontaneous isolated superior mesenteric artery (SMA) dissection: type I, patent true and false lumen revealing entry and re-entry sites; type IIa, patent true and false lumen without re-entry; type IIb, patent true lumen but thrombosed false lumen; type III, SMA occlusion. Reprinted from the *Journal of Vascular Surgery*, 2011; 54:1727-33. Park YJ, Park KB, Kim DI, Do YS, Kim DK, Kim YW. Natural history of spontaneous isolated superior mesenteric artery dissection derived from follow-up after conservative treatment. Copyright 2011, with permission from Elsevier.

systematic review. See the supplemental list of related publications in the Appendix (online only). If multiple reports originated from the same institution and included overlapping time frames, only the most recent report was included. Reports of type B aortic dissection involving the mesenteric vessels and iatrogenic or traumatic dissections were excluded. Fisher exact test was used to compare the failure rate for medical therapy between symptomatic and patients with asymptomatic SMA dissection reported in the literature. An algorithm for treatment at our institution evolved from the literature review and our experience (Fig 2).

Nine patients with MAD (five SMA dissections, three celiac, and one with both) were treated in our practice at the University of Tennessee-Memphis from 2008 to 2013 (Table I). All patients presented with abdominal pain and were started on a medical regimen of anticoagulation, antihypertensive therapy, and serial examination and CTA imaging.

All patients were treated according to the algorithm. Intervention was indicated for persistent symptoms or evidence of malperfusion by CTA imaging. Stenting was considered appropriate for proximal focal lesions. Intimectomy was performed for extensive dissection with compromise of distal branches because this procedure allows restoration of flow to each branch. Surgical bypass was reserved for patients in whom direct inflow could not be obtained. Although no such patients were identified in our series, emergent intervention would be indicated for bowel necrosis or a ruptured aneurysm. Elective treatment of a nonruptured aneurysm would follow established guidelines on the basis of size.

RESULTS

A review of the English literature yielded 143 published reports of spontaneous MAD. Age, sex, vessels involved, symptomatic status, treatment, and results were tabulated. Some reports did not include all of these variables.

Five hundred ninety-six reported cases of MAD (86% men) were identified. Of these, 495 reported cases were SMA dissection (86% men), 88 reported cases were celiac dissection (83% men), and 13 reported cases were combined celiac and SMA dissection (78% men). Eighty-eight percent of all patients were symptomatic at presentation (86% SMA, 95% celiac, 92% both).

Of note, 415 (70%) of all reported patients with MAD originated from China, Japan, or Korea.

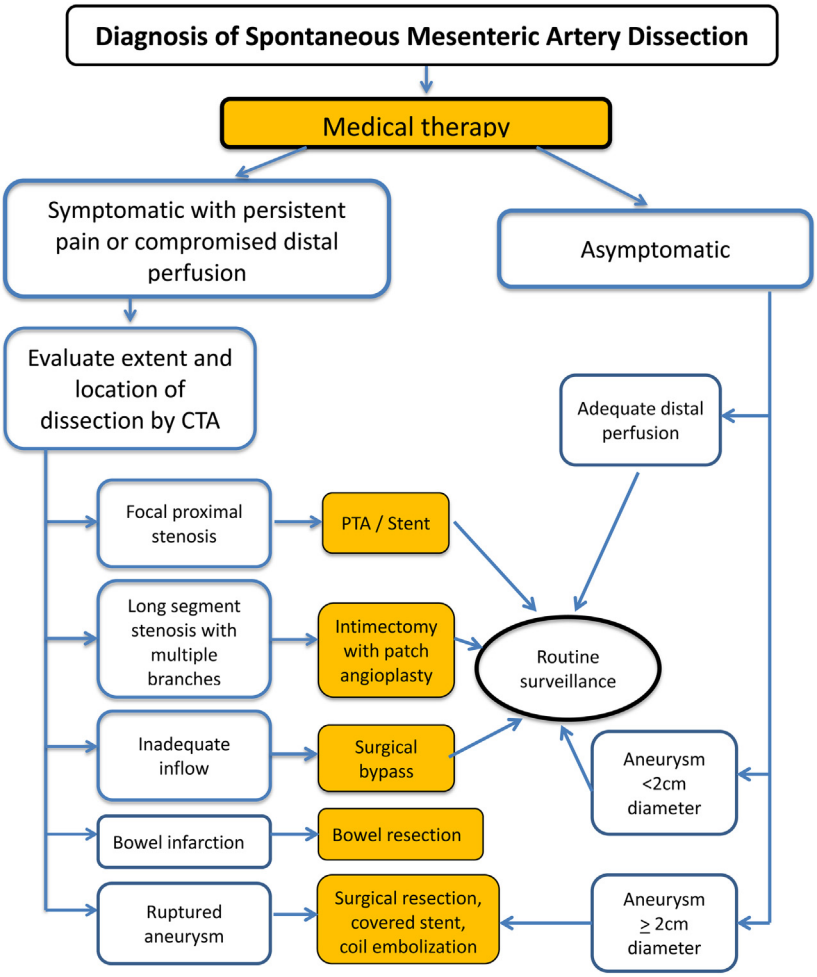

Fig 2. Algorithm for treating mesenteric artery dissection (MAD). *CTA*, Computerized tomographic angiographic imaging; *PTA*, percutaneous transluminal angioplasty.

Regarding patients with SMA dissection, the average age for male patients was 53 years and for female patients 59 years. For 37 patients, the treatment was not reported. Three hundred six patients were treated with medical therapy,[2-17] including some combination of anticoagulation, antihypertensive therapy, and serial examination and CTA imaging. Of these, 41 (13%) failed medical therapy, as indicated by persistent abdominal pain, evidence of bowel ischemia, or development of aneurysmal enlargement. Sixty-seven (22%) patients treated with medical therapy were asymptomatic. All of these were successfully treated with medical therapy except for two patients who required elective treatment for aneurysmal disease.[6] Of the 239 symptomatic patients treated with medical therapy, 39 (16%) failed. Twenty were successfully stented,[6,11,13,14,16,17] although one required a laparotomy for retrograde access to the SMA.[7] Five stents subsequently failed without consequence. Two patients had development of an aneurysm,[2,8] one of which underwent elective surgical repair and one of which was treated with the use of coil embolization for a ruptured SMA aneurysm.[2] Five patients required surgical revascularization, two with the use of bypass and three with the use of intimectomy.[3,9-11] Twelve patients required laparotomy for bowel infarction,[2,5,11-15] one of which required a small-bowel transplant.[12]

Ninety-two patients with SMA dissection underwent primary endovascular therapy chosen by their physician. Eighty-three patients were treated with the use of primary angioplasty and stent,[5,18-23] two of which failed but remained asymptomatic.[18] Ten of these patients were also treated with the use of direct papaverine infusion.[19] Three patients were treated with the use of thrombolytic therapy, all of which failed.[20-22] Two were successfully stented,[20,21] and the other one required surgical intimectomy, which was successful.[22] Two patients had a mesenteric hematoma that did not require intervention. Another patient had a mesenteric abscess that required surgical drainage.[23] Four patients received direct papaverine infusion alone.[19] Two additional patients underwent successful coil embolization for treatment of an aneurysm.[24]

Table I. Algorithm-managed patients 2008 to 2013: Treatment and outcomes

Sex	*Age, years*	*Park class*	*Dissected artery*	*Treatment*	*Outcome*
M	61	III	SMA	Intimectomy and patch angioplasty<24 h after onset of abdominal pain; recurrent thrombosis at 10 days→ redo procedure	Patent SMA by CTA at 48 months. Asymptomatic at 48 months.
M	43	III	SMA	Intimectomy and patch angioplasty 8 days after onset of abdominal pain	Patent SMA by CTA at 50 months. SMA dilated to 24-mm diameter. Asymptomatic at 50 months.
M	46	IIb	SMA	Intimectomy and patch angioplasty 1 day after onset of abdominal pain	Patent SMA by CTA at 60 months. Asymptomatic at 60 months.
F	39	N/A	Celiac	Medical	Stable celiac dissection by CTA at 40 months. Right hepatic branch occluded. Asymptomatic at 40 months.
M	42	IIb	SMA	Medical	SMA dissection resolved by CTA at 19 months. Asymptomatic at 19 months.
F	51	I	Celiac and SMA	Medical	SMA dissection resolved by CTA at 19 months. Splenic and common hepatic occluded near origin but reconstituted. Asymptomatic at 19 months.
M	66	N/A	Celiac	Medical	Celiac artery patent with thrombosed aneurysm by CTA at 16 months. Common hepatic occluded but proper hepatic reconstituted. Asymptomatic at 19 months.
M	82	N/A	Celiac	Medical	Stable celiac dissection by CTA at 6 months. Asymptomatic at 6 months.
F	50	IIb	SMA	Medical	SMA dissection resolved by CTA at 4 months. Asymptomatic at 4 months.

CTA, Computerized tomographic angiographic imaging; *SMA,* superior mesenteric artery.

Fifty-two patients received primary surgical therapy.[5,25-39] Twenty-one underwent surgical bypass,[5,25,26,29,37,38] one of which thrombosed without symptoms and one of which required a subsequent stent.[25,26] One patient required bypass for an aneurysm that developed after intimectomy.[27] Nineteen were treated by use of successful intimectomy.[5,15,28-32] Six required surgical treatment of an SMA aneurysm, two by means of ligation,[33,34] two by means of direct repair,[15] and two by means of bypass,[6,35] which were included in the bypass group. Eight patients required resection of necrotic bowel with no direct intervention for the dissection. Three of these patients died.[36-39]

Eight additional patients died before the diagnosis was made.

Regarding patients with celiac dissection, the average age for female patients was 52 years and for male patients 53 years. For 26 patients, the treatment was not reported. Fifty-one patients were initially treated with the use of medical therapy,[16,40-44] only six of which failed,[16,40-43] four undergoing successful surgical bypass (one for ruptured aneurysm) and two undergoing successful treatment with a stent. Three patients successfully treated with medical therapy continued under observation for celiac aneurysm, and three had splenic infarction. Six patients required primary endovascular therapy, three with stents[40,45-47] and three with coil embolization[48-50] (three for ruptured aneurysms). Five patients were treated surgically,[43,44,51,52] four with the use of bypasses (2 for aneurysm) and one requiring splenectomy for a ruptured aneurysm.[53]

In the small group of 13 patients with combined celiac and SMA dissection, the average age was 53 years. One patient was successfully bypassed.[40] One patient was initially treated with stents that occluded, culminating in successful bypasses.[54] Eleven received medical therapy,[16,17,40,44,55-59] five (45%) of which failed. One of these was treated with the use of a bypass,[16] and four were successfully stented (two for ruptured celiac aneurysm).[17,58,59]

In our series, 67% of patients were male, and the average age was 53 years. All patients were treated medically, and six had resolution of their symptoms. These six patients were maintained on Coumadin anticoagulation for 1 year. Three of four patients with celiac dissection had occlusion of the hepatic artery at an average of 1 year surveillance with no evidence of liver dysfunction. Of six patients with SMA dissection, three required intimectomy for persistent pain during the initial hospitalization (Fig 3). Intimectomy was performed through a longitudinal arteriotomy with individual control of multiple branches. Success is predicated on removal of the dissection flap at the entry site, whereas the dissection end point at each branch may be controlled with tacking sutures. A saphenous vein patch was then applied. There were no perioperative complications. Follow-up CTAs have demonstrated patency of multiple branches with no aneurysmal enlargement at 48 months of surveillance in two patients. One patient with intimectomy has shown gradual dilatation of the SMA diameter from 19 to 24 mm over 4 years. All patients remain asymptomatic, with no recurrent symptoms or mortality.

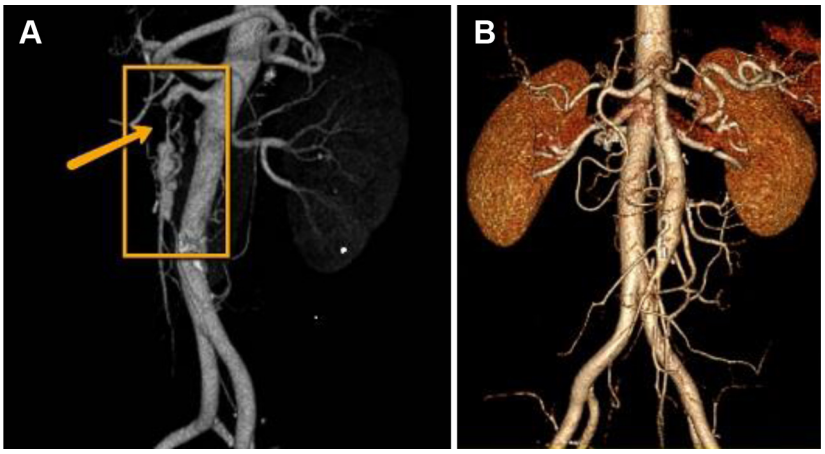

Fig 3. A, Preoperative computerized tomographic arterial (CTA) imaging shows superior mesenteric artery (SMA) dissection. **B,** Patent SMA on CTA imaging 4 years after intimectomy.

Table II. Summary of systematic literature review

Dissected mesenteric artery	*SMA*	*Celiac*	*Both*
Average age, years	54	53	53
Men, %	86	83	78
Treated medically	306	51	11
Asymptomatic at presentation, No. (%)	67 (22%)	2 (4%)	0 (0%)
Asymptomatic patients who failed medical treatment, No. (%)	2 (3%)	0 (0%)	N/A
Symptomatic patients who failed medical treatment, No. (%)	39 (16%)	6 (12%)	5 (45%)

SMA, Superior mesenteric artery.

DISCUSSION

Patients with MAD represent a diverse group. Indications for intervention include risk of bowel ischemia, aneurysmal enlargement, or rupture. For this reason, the literature can be confusing regarding time of observation and methods of intervention if necessary. Multiple algorithms for treatment have been published, but none include all aspects of pathology and treatment. This systematic review was undertaken as an attempt to bring some clarity to the decision process.

After the systematic review of the literature and our own experience, it remains difficult to determine which patients can be treated with the use of medical therapy if symptoms do not rapidly resolve. Medical therapy is generally successful in the patient with asymptomatic MAD or the patient with a patent dual-lumen artery in the absence of aneurysm formation. Medical therapy may be successful in a majority of other patients with MAD as long as the compromised lumen is adequate and/or adequate collaterals exist. However, although medical therapy is successful in 86% of patients, a substantial number continue to have symptoms, occasionally resulting in bowel infarction. Therefore, careful observation and surveillance is indicated; each patient must be assessed on an individual basis, taking into account both anatomy and symptoms. The overwhelming majority of patients with SMA dissection who failed medical therapy required intervention primarily for persistent pain. Of the 17 patients with celiac dissection who required intervention, seven (40%) required treatment of an aneurysm, and three patients remained under aneurysm surveillance after successful medical therapy. It is not surprising that 45% of patients with both SMA and celiac dissection failed medical therapy because more collateral circulation was at risk.

Symptomatic status was evaluated as a predictor of successful medical therapy in patients with spontaneous MAD. Among patients treated with medical therapy, a two-tailed Fisher exact test was used to compare the failure rate for medical therapy between patients with SMA dissection who presented with and without symptoms. Asymptomatic status was a statistically significant predictor of successful medical therapy for SMA dissection ($P = .0037$). There were insufficient numbers to detect a difference in patients with dissection involving the celiac or both mesenteric vessels (Table II). Likewise, there was insufficient anatomic data in the literature to perform a statistical evaluation.

If intervention is indicated, the choices are largely based on anatomic considerations. Although intimectomy has some advantage for preserving branch flow, stenting of the true lumen is often successful and likely sufficient if the patient is a poor risk for open revascularization. The long-term patency for stents in this location for this disease, however, is unknown. The long-term success of intimectomy may be dependent on the integrity of the remaining vessel wall. One of 20 patients in the literature had development of aneurysmal enlargement after intimectomy, and one of our patients has demonstrated enlargement to 24-mm diameter. Few centers have experience with papaverine infusion, which, nevertheless, may prove to be a beneficial adjunct to medical or interventional therapy. Treatment of aneurysms may include surgical interposition grafting, stenting, or coil embolization, depending on the anatomy of the aneurysm.

In patients who became asymptomatic after medical therapy, most centers have continued CTA imaging every 6 months for 1 year and then yearly in the asymptomatic patient. Continued antithrombotic therapy for 6 to 12 months has been used in random fashion. If the patient remains asymptomatic with a patent target mesenteric vessel at 1 year with discontinuation of antithrombotic therapy, further surveillance may only be indicated for evidence of aneurysmal enlargement. No information regarding delayed aneurysmal enlargement could be obtained from the literature, given the very short follow-up reported. In addition, no information regarding a definitive surveillance or outpatient antithrombotic protocol could be gleaned from the literature review. The risk of aneurysmal enlargement over the long term is an important topic for future investigation.

CONCLUSIONS

All available treatment options for MAD are adequate in the appropriate circumstances. The algorithm described may be helpful for guiding treatment.

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AUTHOR CONTRIBUTIONS

Conception and design: HG
Analysis and interpretation: HG
Data collection: HG
Writing the article: HG
Critical revision of the article: HG
Final approval of the article: HG
Statistical analysis: HG
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Overall responsibility: HG

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Additional material for this article may be found online at www.jvascsurg.org.

APPENDIX (online only). List of related publications.

1. Ahn S, Sohn CH, Seo DW, Lim KS, Kim W. Diagnosis of spontaneous isolated superior mesenteric artery dissection in the emergency department: a case series. J Korean Soc Emerg Med 2010;21:615-21.
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